EXHIBIT R

Northwell Health

North Shore-LIJ Health System is now **Northwell Health**

Occupational Medicine, Epidemiology and Prevention

March 4, 2016

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Simon, Greenstone, Panatier, Bartlett
3232 McKinney Avenue
Suite 610
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Re: Valeria Dalis

Dear Ms. Kagan

I am writing to report the results of my evaluation of the materials listed below pertaining to Ms. Valeria Dalis. I have reviewed these materials in the context of my pre-existing knowledge, training, and experience in the field of occupational medicine. These materials are of the type I and other specialists in occupational medicine normally rely upon and are sufficient to form a reliable basis for my opinions contained within this report. All of the opinions stated in this report are given within a reasonable degree of medical certainty.

This report and the opinions stated in the report are based on the listed materials and my 24 years of training, education, and experience in the area of asbestos-related occupational medicine. Over the past 24 plus years, I have had the opportunity to evaluate and treat hundreds of patients with asbestos exposure, many of whom have asbestos related diseases.

Qualifications:

I am a physician licensed in the State of New York, specializing in the field of occupational and environmental disease. I have been a practicing physician since I graduated from medical school in 1988. A copy of my Curriculum Vitae, dated 3/2/16 is attached to the report as Exhibit 1.

I attended the University of Chicago and received a Bachelor of Arts degree with Honors, with a major of History, Philosophy and Social Studies of Science and Medicine. I then continued at the University of Chicago – Pritzker School of Medicine, where I obtained my medical degree in 1988. I was elected to the Alpha Omega Alpha Honor Society, and was also awarded an American Medical Women's Association Award. Following medical school graduation, I was an intern and resident in Internal Medicine at Yale University – Yale New Haven Hospital from 1988 – 1991. Upon completion of my Internal Medicine Residency program, I completed a second residency at the Mount Sinai School of Medicine in Occupational Medicine, from 1991 – 1993. During my Occupational Medicine Residency Program, I obtained my Master of Science Degree in Community Medicine (equivalent degree to a Masters of Public Health) in 1993. I began to evaluate dozens of patients with asbestos exposure during my residency program at Mount Sinai. I am board certified in Occupational Medicine and in Internal Medicine. I have become recertified in Internal Medicine two times.

Following completion of my residency training in Occupational Medicine, I was awarded a Fellowship in Occupational Medicine from the Foundation for Occupational Health and Research. I continued at Mount Sinai, where I joined the faculty, and continued to evaluate patients with asbestos exposure. I became Vice Chair of the Department of Preventive Medicine in 2001. I was Director of the New York/New Jersey Education and Research Center from 2006 – 2010, and had been Director of the Residency Program in Occupational Medicine from 1998-2006. I was also the Director of the Mount Sinai World Trade Center Medical Monitoring and Treatment Program from 2006 – 2010, although my involvement with the World Trade Center medical programs started in 2001, when I began to evaluate patients with exposure to the World Trade Center disaster, and was initially Medical Core Director of the World Trade Center Worker and Volunteer Medical Screening Program (2002-2004), and Co-Director of the World Trade Center Medical Monitoring and Treatment Program (2004-2006). I have published over fifty articles in the peer-reviewed literature.

In 2010 I left the Mount Sinai School of Medicine to become the Founding Chair of the Department of Population Health at Northwell Health and Hofstra Northwell School of Medicine (formerly known as North Shore University Health System). The Department changed its name in 2014 to Occupational Medicine, Epidemiology and Prevention.

I have evaluated hundreds of patients with asbestos exposure in my career in occupational medicine, spanning nearly 25 years. I currently direct the Occupational and Environmental Medicine Center of Long Island, providing occupational health services to

patients in the metropolitan New York area. Over the past year alone, I have supervised the examination of or directly examined nearly 500 patients with asbestos exposure, as we have greatly expanded our clinical services. Over the course of the past 25 years, I have evaluated dozens of patients with malignant mesothelioma and lung cancer due to asbestos exposure. I have kept abreast of the scientific and medical literature regarding the diagnosis and causation of mesothelioma. I have personally evaluated cases of mesothelioma where the exposure was brief, and have also seen cases of mesothelioma in individuals whose only exposure to asbestos was from family members who worked with asbestos and brought their asbestos contaminated clothes home.

Materials Reviewed:

I have had the opportunity to review the medical records and deposition transcripts of Ms. Dalis I was provided with the following information:

1. Crisis Life Center, medical records
2. Dr. Duraga V. Madala, medical records
3. El Camino Hospital, medical records
4. Dr. Forouzan Vaghar, medical records
5. Foundation Medicine, medical records
6. Good Samaritan, medical records
7. Dr. Mohammadreza Rohaninejad, medical records
8. Dr. Mojgan Morshedi, medical records
9. National Surgical Associates, medical records
10. Palo Alto Medical Foundation, medical records
11. San Jose Cardiac Surgical, medical records
12. Stanford Hospital, medical records
13. UCSF, medical records
14. Valley Medical Oncology, medical records
15. Valley Radiology Imaging, medical records
16. Plaintiffs' Certified Amended Answers to Part I and Part II Interrogatories
17. *de bene esse* Deposition of Valerie Dalis
18. Discovery Deposition of Valerie Dalis
19. Discovery Deposition of Nicholas Dalis
20. Dr. Maddox Report
21. Ronald Gordon, Ph.D., expert report, dated March 1, 2016
22. Sean Fitzgerald, PG, expert report, dated February 25, 2016
23. Company product testing documents
24. Cosmetic Toiletry and Fragrance Association documents
25. August 3, 1972 S. Lewin to A. Weissler of Food and Drug Administration
26. May 8, 1973 Proceedings of the Symposium on Talc, Washington DC by A Langer
27. July 31, 1973 Memorandum, A. Weissler of Food and Drug Administration re Summary and Comments on Prof. Lewin's Analytical Results for Asbestos in Talc

Ms. Dalis's Medical and Exposure History:

Clinical History: Ms. Dalis had a CT scan of the abdomen and pelvis on August 19, 2014 that showed omental caking, a large volume of ascites, with a fluid pocket evident in the right lower quadrant, and enlarged diaphragmatic lymph nodes. There was no pancreatic or gastric mass noted. On August 20, 2014, Ms. Dalis underwent a paracentesis, with approximately 2,800 cubic centimeters of gelatinous fluid removed from the abdomen. An omental biopsy was also performed. The cytology from the peritoneal fluid showed "atypical mesothelial cells", which were reactive versus malignant. The omental biopsy showed benign small intestinal mucosa and fragments of smooth muscle and was not diagnostic.

Ms. Dalis underwent a laparotomy, appendectomy, omentectomy and left salpingo-oophorectomy on August 25, 2014. Dr. Rohaninejad described possible pseudomyxoma peritonei. The pathology showed malignant mesothelioma, epithelioid cell type. The pathology was sent for additional review at UCSF and Stanford Pathology. The pathologists agreed with the diagnosis of malignant mesothelioma. Dr. Maddox also reviewed the pathology and agreed with the diagnosis of malignant mesothelioma, epithelioid cell type of the peritoneum.

Ms. Dalis went to see Dr. George Labban, a medical oncologist, on September 3, 2014. She had been complaining of abdominal pain for the prior two months. She had weight loss, nausea, heartburn, depression, anxiety and decreased sex drive. Her pain had improved since the surgery. Dr. Labban ordered a PET/CT scan for staging and to rule out extra abdominal disease. Treatment options were discussed, and depended on the extent of the malignancy.

A mammogram on September 9, 2014 was negative for malignancy. A PET/CT scan on September 11, 2014 showed increased uptake in the abdomen consistent with malignant ascites, omental metastatic disease, and cardiophrenic nodules suspicious for malignancy. There were hypermetabolic nodules in the right lung, post-surgical changes in the anterior abdominal wall and a possible abscess in the right lower abdomen. Dr. Labban saw her on September 17, 2014. Dr. Labban planned to present her case at the Tumor Board later that week, and treatment decisions regarding chemotherapy or surgery would be discussed. Dr. George Fisher at Stanford saw Ms. Dalis on September 24, 2014. He noted that her case had been presented at the GI Tumor Board at Stanford, and recommended more standard chemotherapy with Cisplatin and Alimta, while noting HIPEC was an option. He also thought genomic testing of the tumor might be useful, and that he would see her for consideration of clinical trials if the chemotherapy were ineffective. Ms. Dalis went to see Dr. J. Augusto Bastidas, a gastrointestinal surgeon, on September 26, 2014 for a second opinion regarding treatment options. Dr. Bastidas discussed with Ms. Dalis and her husband that there was no basis for regional therapy with cytoreductive (CRC) surgery and HIPEC for the abdomen given the likelihood that

there was extra-abdominal disease. He recommended that she see Dr. Silva to discussion options for the resection/biopsy of the nodes/nodules seen on the CT that appeared to be in the thorax. If those lesions were not malignant, then CRC/HIPEC could be reconsidered. Dr. Labban also saw Ms. Dalis on September 26, 2014 at an office visit. He noted that her case had also been presented at the El Camino Hospital tumor board, and the consensus opinion was to proceed with the biopsy of the cardiophrenic soft tissue nodule to rule out or rule in metastatic disease. If the biopsy were positive, then she would be considered for systemic chemotherapy. If it were negative, then she would consider HIPEC and cytoreductive surgery.

Dr. Raymond Silva, a thoracic surgeon, saw Ms. Dalis on October 3, 2015 because of the finding of the soft tissue densities in the cardiophrenic fat pad, and the potential treatment implications if the tumor had indeed spread the thorax. At the time of the visit, Ms. Dalis noted fatigue and depression, and Dr. Silva noted that she was considering the hyperthermic chemotherapy option, if it were possible. He planned to perform a thoracoscopy on October 8, 2014.

On October 8, 2014, Ms. Dalis underwent a right video-assisted thoracoscopy and biopsy of the diaphragmatic nodules. At the time of surgery, Dr. Silva noted that the right hemidiaphragm was “strewn in its entirety with firm nodules.” The biopsy material from the nodules showed malignant mesothelioma. While she was in the hospital after the thoracic surgery, she also had a paracentesis, with 780 cubic centimeters of fluid removed from the abdomen. She was discharged home on October 10, 2014.

Ms. Dalis went to the Emergency Department at Good Samaritan Hospital on October 13, 2014 with complaints of weakness, fever and blood tinged sputum since the thoracoscopy on October 8th. A chest x-ray showed a small right pleural effusion with compressive atelectasis or consolidation in the right lung base. The effusion was slightly increased compared to October 10th.

Ms. Dalis went to see Dr. Labban on October 15, 2014. She was on antibiotics for bronchitis/pneumonia and had an improvement in her chest congestion, fever and coughing. She complained of abdominal bloating and discomfort. Dr. Labban planned to treat her with combination chemotherapy with Alimta and Pemetrexed because of the spread of tumor into the thoracic cavity. She had a paracentesis on October 17, 2014, with 1,150 cubic centimeters of fluid removed from the abdomen.

Dr. Rohaninejad inserted a Mediport on October 24, 2014 for systemic chemotherapy. Dr. Labban saw Ms. Dalis on November 3, 2014. She had alternative diarrhea and constipation, but her bowel movements had stabilized. She had fatigue and abdominal wall pain. He noted that she had had a recent echocardiogram after a chest x-ray showed cardiomegaly. A slight pericardial effusion was noted, along with normal left ventricular ejection fraction of 55% and normal function. Dr. Labban started chemotherapy with Alimta and Cisplatin on November 4, 2014. Ms. Dalis returned to Dr. Labban on December 1, 2014 for her second cycle of Alimta and Cisplatin. Dr. Labban saw Ms. Dalis on December 9, 2014. She complained of mouth sores, fever, fatigue,

nausea and a decreased oral intake. Dr. Labban treated her for dehydration with intravenous fluids, and noted that she had mucositis from the Alimta. He recommended magic mouthwash.

Dr. Labban treated Ms. Dalis on December 22, 2014. She noted the hydration made her feel much better with less fatigue. Her mouth sores had also resolved after increasing the dose of folic acid. Ms. Dalis was anemic and he treated her with chemotherapy, and she also received a blood transfusion with two units of packed red blood cells on December 22, 2014. Ms. Dalis returned to Dr. Labban on January 12, 2015 for chemotherapy. Dr. Labban planned a PET/CT scan after her chemotherapy to assess progress.

A PET/CT scan on January 28, 2015 showed a resolution of multiple hypermetabolic soft tissue lesions within the peritoneum, particularly within the pelvis. However, there had been an overall increase in the large volume ascites within the abdomen and pelvis with persistent FDG activity, consistent with malignant ascites. There were continued areas of soft tissue nodularity within the peritoneum with associated mild FDG activity. There were multiple previously identified hypermetabolic abdominal wall lesions that were stable and slightly increased in size, but had decreased FDG activity. There was persistent mild diaphragmatic lymphadenopathy, with maximal SUVs stable to slightly decreased. No new lymphadenopathy was seen, and the nodular densities in the right lung were not FDG avid and stable in size.

Dr. Labban saw Ms. Dalis on February 2, 2015. She was complaining of a frontal headache that had persisted for two weeks. Dr. Labban noted that her ascites had re-accumulated and she complained of generalized weakness. He planned an MRI of the brain because of the headaches, and planned for continued Cisplatin and Pemetrexed after the paracentesis. Dr. Morshedi saw her on February 3, 2015 to check her blood pressure, and started her on Amlodipine tablets.

Ms. Dalis had a paracentesis on February 5, 2005 with 3,700 cubic centimeters of fluid removed. Dr. Labban evaluated her on February 9, 2015 and noted that she had felt much better after the last paracentesis. She received her fifth cycle of combination chemotherapy. She had an attempted paracentesis on February 25th but it was terminated without removal of fluid due to pain. Ms. Dalis saw Dr. Labban on March 2, 2015. She continued to have fatigue and abdominal distension. She received her sixth dose of Alimta and Pemetrexed on March 3, 2015.

A paracentesis was done on March 25, 2015, with 3,000 cubic centimeters of fluid removed from the abdomen. A PET/CT scan on April 14, 2015 showed stable intra-abdominal and abdominal wall disease and stable anterior diaphragmatic lymphadenopathy, with the abdominal wall lesions appearing more well-circumscribed than on previous examinations. Thickening of the peritoneum along the diaphragm was also without significant change. There was no evidence of progression and no new nodules were noted. Ms. Dalis returned to Dr. Labban on April 21, 2015. Dr. Labban noted that her recent scans were stable. She was to see if she were eligible for a clinical

trial at Stanford, and whether she should remain on single agent Pemetrexed. Dr. Fisher saw Ms. Dalis on May 4, 2015. He recommended second line chemotherapy with Gemcitabine, Gemcitabine and Taxane or Taxane alone. He also discussed experimental therapies. Dr. Labban saw Ms. Dalis on May 5, 2015. She decided to start Gemcitabine. Ms. Dalis had a paracentesis on May 7, 2015, with 4,000 cubic centimeters of fluid removed from her abdomen. At a return visit to Dr. Labban on May 14th, Ms. Dalis had leukopenia and neutropenia. He planned to decrease the Gemcitabine dose and postpone chemotherapy for one week, and start Neulasta. Ms. Dalis returned for chemotherapy with Gemcitabine on May 8th with Neulasta on day 2 following the chemotherapy. Ms. Dalis returned to Dr. Labban on June 1st. She had less ascites but a local fever in her abdominal wall and the mesothelioma metastatic lesions. She received the Gemcitabine, and Dr. Labban planned to lower the dose of the Neulasta since Ms. Dalis had a reaction.

Dr. Labban saw Ms. Dalis on June 8, 2015. She was on single agent Gemcitabine, and complaining of fatigue and a nodule in her abdominal wall. Dr. Labban palpated three nodules in her abdomen, one in her right lower quadrant and two in the left upper quadrant. She received Gemcitabine with a reduced dose of Neulasta due to the side effects from the prior dose. Dr. Labban saw Ms. Dalis on June 22nd. She started her third cycle of Gemcitabine. Ms. Dalis underwent a paracentesis on June 24, 2015, with 3,300 cubic centimeters of fluid removed. Ms. Dalis returned to Dr. Labban on June 29, 2015. She had fatigue and noted a “lower temperature” in the abdominal wall masses. She received systemic chemotherapy with Gemcitabine, and she received two units of packed red blood cells at Good Samaritan Hospital on July 1, 2015. Dr. Labban ordered genetic profiling from Caris to see if there was a molecular profile that would expand the treatment options. A PET/CT scan on July 7, 2015 showed stable appearance of mildly hypermetabolic anterior diaphragmatic lymphadenopathy. There was stable large volume abdominal/pelvic ascites with stable mildly hypermetabolic soft tissue nodularity of the omentum. There was a mild increase in the size of the bulky mildly hypermetabolic anterior abdominal wall lesions, left greater than right, with similar low level metabolic uptake compared to the prior study. Ms. Dalis returned to Dr. Labban on July 13, 2015. She continued to have fatigue and abdominal bloating, but denied any pain. Dr. Labban was awaiting the results of the genetic profile from Caris (Foundation One had not proved to be practically useful). He noted that there was mixed response to the Gemcitabine and planned to continue while awaiting the genetic profile from Caris.

Ms. Dalis went to see Dr. Fisher on July 28, 2015. He noted that Ms. Dalis had been on single agent Gemcitabine in May and June, and that her last dose was June 10th, for a total of two cycles. The PET scan showed progression of disease, and Ms. Dalis was having difficulty with fatigue and required frequent paracentesis. Dr. Fisher noted that she now required transfusions and that might limit her ability to tolerate Taxol. In general, Dr. Fisher noted that there were few medications that were shown to be useful; he also discussed the potential for debulking the tumor, although it was usually done in individuals with minimal or no ascites. He suggested Xeloda, and if she had a response to the Xeloda, then surgery might be an option.

Dr. Labban saw Ms. Dalis on July 30, 2015. She continued to have abdominal distension and bloating. A paracentesis was planned for the next day. Dr. Labban noted that she was progressing on Gemcitabine, and they reviewed the genetic analysis with potential benefit from chemotherapy with oral Temozolomide in addition to other agents, and while it was not FDA approved for mesothelioma, they planned to proceed based on the genetic analysis of her tumor. A paracentesis was done on July 31, 2015 with 3,500 cubic centimeters of fluid removed.

Ms. Dalis started Temozolomide on August 10, 2015. She returned to Dr. Labban on August 18th noting increased swelling and discomfort in her abdominal wall due to the growing abdominal wall masses. She had fatigue. Dr. Rohaninejad saw Ms. Dalis on August 25, 2015 for a surgical consultation. Ms. Dalis had noted that the abdominal wall tumor had increased in size and was getting very uncomfortable, and sought Dr. Rohaninejad's opinion regarding palliative debulking. He felt that she should continue chemotherapy and that the tumor was possibly growing at her port sites. He planned to discuss possible surgery with Dr. Labban.

Ms. Dalis had increasing abdominal pain and growth in the abdominal masses. She had a paracentesis on September 4, 2015 with 3,600 cubic centimeters of fluid removed from the abdomen. She returned to Dr. Labban on September 30, 2015. She was having nausea and requested pain medication for the abdominal discomfort. She had chronic constipation and increasing and progressive fatigue. He planned to treat her with Abraxane for two to three cycles, because the genetic profiling showed a possible response; there had been no response to the temozolomide. A paracentesis on October 1, 2015 was done, and Ms. Dalis had 4,800 cubic centimeters of fluid removed from her abdomen. Ms. Dalis started Abraxane for fourth line treatment on October 2, 2015. Dr. Labban saw Ms. Dalis on October 19th. She had severe fatigue and joint pain that she attributes to the chemotherapy and the Neulasta. He abdominal pain and distension had progressed, and she felt that tumor in her abdomen was pressing on her stomach and making her short of breath. Ms. Dalis noted that the Tramadol was not controlling her pain and Dr. Labban prescribed Norco for pain. He referred her to radiation oncologist because the tumors were bulky and causing pain and discomfort. He also reminded her to follow-up with Dr. Fisher for a second opinion. Ms. Dalis wanted to continue the Abraxane and Neulasta, but at a lower dose.

Ms. Dalis went to see Dr. Gordon Wong on October 21, 2015. He noted that she had multiple paracentesis, with her most recent procedure earlier that week on October 20th, where 3,300 cubic centimeters of fluid was removed. She was taking Abraxane, with the next dose scheduled for October 27th. Dr. Wong evaluated Ms. Dalis for the potential role of radiation therapy in treatment of her mesothelioma. At the time of her evaluation, she had decreased appetite and worsening fatigue. She had nausea and diarrhea after her last treatment with Abraxane, which had resolved, but noted a flare in her abdominal wall lesions and joint pain that lasted 1-2 weeks after the last treatment. Dr. Wong discussed potential radiation for her abdominal wall lesions but noted that radiation treatments might complicate any abdominal surgery. She was to see Dr. Fisher at Stanford regarding possible surgery, and Dr. Wong planned to coordinated treatment after receiving

recommendations from Dr. Fisher. Dr. Fisher saw Ms. Dalis on November 9, 2015. At this point, Ms. Dalis was spending most of her time in bed due to pain from the tumors, and was fatigued with a poor appetite. She did not like the way narcotics made her feel and was taking Tramadol for pain. Dr. Fisher noted that Ms. Dalis's tumor had been resistant to multiple chemotherapy agents, and she was not a surgical candidate due to the volume of her disease burden in addition to the diffuse sits and rapid recurrence post-surgery in July 2014. He recommended targeted radiation per Dr. Wong's recommendations and considering a permanent catheter to drain the abdominal fluid. Dr. Fisher also felt that if she did not receive significant relief from the Abraxane after two cycles and it continued to cause the significant side effects, it should be discontinued.

Ms. Dalis continued to have reaccumulation of the peritoneal ascites, and underwent paracentesis on November 11th (3,750 cubic centimeters) and 19th, 2015 (2,500 cubic centimeters). Dr. Labban saw Ms. Dalis on November 17, 2015. She was having abdominal bloating and discomfort. Dr. Labban noted that Dr. Fisher had discussed hospice care with Ms. Dalis. Dr. Labban also discussed hospice and consulted with Dr. Wong for consideration of palliative radiation. He discontinued the Abraxane due to intolerance. Ms. Dalis requested a paracentesis for discomfort. Dr. Wong saw Ms. Dalis on November 23, 2015. She related that she had had treatment with Abraxane on October 27th, 2015 and had significant side effects and was essentially bedbound. Dr. Fisher did not recommend surgery, and recommended consideration of radiation as well as hospice. Ms. Dalis had pain that ranged from 6-9/10 in the abdomen and also had constipation. She was easily fatigued and had exhaustion and dyspnea at less than one flight of stairs. Dr. Wong noted that Ms. Dalis was not going to undergo additional chemotherapy at this time due to her intolerance. He planned to treat her with palliative radiation aimed at the bulky mesothelioma lesions over the course of three weeks.

Ms. Dalis continued to have fluid accumulation in her abdomen, and underwent a paracentesis on December 9, 2015 with 2,500 cubic centimeters of fluid removed from the abdomen. Ms. Dalis had a paracentesis on December 26, 2015, with 3,300 cubic centimeters of fluid removed from the abdomen. An ultrasound on December 31, 2015 was performed in preparation for a paracentesis. There was some ascites, but no pockets of fluid were identified that would be large enough to tap, so no paracentesis was performed.

No additional medical records were available for my review.

Ms. Dalis passed away on February 21, 2016. She was 68 years old.

Past Medical History: Ms. Dalis had a history of hypertension, depression, kidney stones and had an oophorectomy for a benign ovarian cyst. She has had a hernia repair, a C-section and cataract surgery.

Cigarette Smoking History: Ms. Dalis smoked cigarettes in the 1970s for around 18 months.

Occupational and Environmental History: Ms. Dalis grew up in a home where her mother used Cashmere Bouquet talcum powder for "as long as [she could] remember." Ms. Dalis recalls that her mother applied Cashmere Bouquet powder onto her body using a powder puff, and that there would be powder all over the place. She was in the bathroom with her mother at times when her mother applied the talcum powder onto her body. She could see the talcum powder falling onto a dark surface. Ms. Dalis recalled personally using the Cashmere Bouquet talcum powder starting when she was around nine or ten, and applying the powder with the puff to her armpits, groin and around her body. She used it throughout her pre-teen and teenager years while she was living with her mother, apart from an approximate 18-month period when she lived with her father. Ms. Dalis noted that her mother used to refill the powder puff from the shaker bottle, and that her aunt also used Cashmere Bouquet.

When Ms. Dalis moved out of her mother's home when she was 18, she continued to use Cashmere Bouquet, applying it either with a powder puff or via the shaker. She recalled that there was powder all over the place, which she would have to clean up. Ms. Dalis continued to use Cashmere Bouquet until the 1990s, when she noted that the powder was visible on the dark marble in her bathroom.

Ms. Dalis had additional exposure to talcum powder when working as a licensed cosmetologist in California. She completed cosmetology school in 1966 and began working at a hair salon in downtown San Jose, cutting hair, doing hair color and applying permanent waves. She switched to a second salon in Willow Glen after about one year, and worked there for one year as well. In the late 1960s Ms. Dalis went to work at Gere's Casa de Bellesa, a salon, where she worked for about two to three years. In the salons, Ms. Dalis used Mennen's talcum powder after she used clippers on the neck during short haircuts. She stated that she used the powder on the neck on a daily basis while working in the hair salons. She shook the powder onto the necks and wiped the powder off with a brush or blow dryer. She also applied talcum powder to a brush and then applied it to the neck. In addition, Ms. Dalis described using Mennen's talcum powder in the plastic gloves, which was necessary in order for her to get the gloves onto her hands. She described wearing gloves on a regular basis to apply color, and she had to blow the powder into the gloves.

Ms. Dalis worked in a clothing boutique for several years in the 1970s and then stopped working after she married Mr. Dalis in the early 1970s. She occasionally worked in interior design, choosing color schemes and fabrics in the 1980s. Ms. Dalis and her husband built a home on Douglas Lane from 1985-1995, and remodeled the kitchen and family room when they moved to Saratoga in the 1990s.

Conclusion: Ms. Dalis suffered and died from malignant mesothelioma of the peritoneum with spread to the chest as a result of her exposures to asbestos from cosmetic talc. She underwent two surgeries as well as multiple rounds of chemotherapy. Eventually, she succumbed from the mesothelioma.

Based on the information available, it is my opinion, to a reasonable degree of medical certainty that Ms. Dalis's exposure to asbestos-containing talcum powder led to the development of her mesothelioma. She began using Cashmere Bouquet in the 1950s and continued to use it daily for decades. She had additional workplace exposure to Mennen Powder in the late 1960s while working as a hairdresser.

The methodology and basis for my opinions follows standard methods of the medical and scientific community. Asbestos is the most well known cause of mesothelioma, and the causation of mesothelioma has been established by the quantitative history of exposure to asbestos. Thousands of individuals, from myriad professions and exposure situations have developed mesothelioma as a result of either direct or indirect exposure to asbestos. The reliance on the history of exposure to asbestos was used by seminal studies by Newhouse, Wagner and Selikoff in the 1960s, who attributed mesothelioma to asbestos exposure based solely on the history of exposure. The increased risks for mesothelioma exist for individuals who both worked directly with asbestos products and for those who worked adjacent to or in the vicinity of others who were using asbestos products, which is known as "bystander" exposure.

<u>Asbestos and Malignant Mesothelioma General Opinions:</u> Occupational Medicine is the field of medicine that deals with exposures to substances, toxins, conditions and agents in the workplace that are associated with increased risks of diseases. It exists as a subspecialty of Preventive Medicine that deals with identifying ways to prevent people from becoming ill. This includes identifying the sources, agents or catalysts that increase the likelihood of someone developing a disease, illness, or detrimental condition, and educating people on how to eliminate, avoid, and/or mitigate those risks. To put it simply, Occupational Medicine and Preventive Medicine involves searching for and identifying causes of diseases. This knowledge is important for those who are already ill: elimination of the catalysts can eliminate or mitigate the illness. It is also important from a public health point of view: to a large extent, the higher purpose of Occupational Medicine and Preventive Medicine is to educate and warn the public on how to eliminate, avoid, or mitigate the risks of diseases at the workplace, and to provide guidance to governments and businesses on appropriate regulations and standards concerning workplace health and safety.

One of the essential tasks of a physician of Occupational Medicine, when dealing with an individual patient, is the taking of a proper occupational history. Standard medical histories usually involve the patient explaining their reason for seeking medical attention; a listing of current symptoms, conditions, allergies, medications and other relevant medical problems; and providing some family and social history. Occasionally, a standard medical history may-but doesn't always-include identifying the patient's occupation.

A full occupational history, on the other hand, will go into details of a patient's entire work history, including details concerning their tasks and duties and their working conditions and environment. The history will also routinely make inquiries into the patient's home or hobbies. It would also reveal what kinds of substances or agents the

patient was exposed to in his or her working environment that might have occurred decades earlier. It remains the standard tool for determining exposure and has not be supplanted by quantitative measurements, which are rarely obtained, and would not, unless continuously performed on an individual (which is not feasible), fully address all exposures an individual might have had. At times, it is not possible to directly obtain an occupational history from an individual, and information concerning work and environmental experiences contained in deposition transcripts by plaintiffs, co-workers and family members can provide detailed information of that type that can be elicited from an occupational physician-obtained history.

The hallmark of occupational medicine is to connect an exposure to a hazardous substance to a disease, and identify whether there is a causal relationship. This is a critical differentiation in the field of occupational medicine; not only do we treat patients for disease, but we emphasis what hazardous substance might be causing the disease. In occupational medicine training, there are core areas of training, including epidemiology, biostatistics, toxicology, and industrial hygiene.

Asbestos and Disease: Asbestos is a naturally occurring mineral that has been used commercially for a variety of purposes for over 100 years. Asbestos is mined in the form of microscopic fibers released from the surrounding earth. Asbestos was extremely useful from an industrial perspective: it is highly resistant to heat and therefore serves as an excellent insulator and friction surface. It is also very durable, and as a fiber it can be molded into shapes and products that serve a variety of functions. However, asbestos is also highly toxic and carcinogenic when the fibers are inhaled or ingested.

While there are many "fiber types" of asbestos, as well as different sizes of the fibers, there exists consensus among scientists that exposure to *any* asbestos fiber type or size increases the likelihood of lung cancer, mesothelioma, as well as nonmalignant lung and pleural disorders. Asbestos fibers are generally divided into two categories: amphiboles and serpentine (or chrysotile). There are several varieties of amphiboles, including both commercial and non-commercial types. The three major asbestos types used in industry have been chrysotile, amosite and crocidolite. Of these three fiber types, over 95% of all asbestos used in the United States has been chrysotile. Much of the chrysotile asbestos that was used in the US was mined in Canada, where there was contamination with small amounts of tremolite, another type of amphibole asbestos. The mainstream scientific community has also long recognized, and continues to recognize today, that there is no "safe" level of exposure to asbestos regardless of fiber type or size. This position is shared by numerous United States government agencies, including the Occupational Safety and Health Administration ("OSHA", which has regulatory authority over workplaces), the Environmental Protection Agency ("EPA" which has regulatory authority over non-occupational settings), the National Institute for Occupational Safety and Health ("NIOSH", which is responsible for conducting research and making recommendations for the prevention of work-related injuries and illnesses), the World Trade Organization ("WTO"), and the national academies of science of every major industrialized nation. The World Health Organization recently reviewed the existing literature and concluded (in 2014) that all fiber types are capable of causing asbestos

related disease, including mesothelioma, and reiterated the statement that there is no safe level for exposure to asbestos.

Due to the ubiquitous use of asbestos and its presence in naturally occurring formations, there is asbestos in the ambient air in the United States, albeit at minute levels. The ambient air concentration or "background level" has been reported to range from 0.0005 f/cc in urban areas, to 0.00005 f/cc in rural regions. These levels are thousands of times less than the current OSHA permissible exposure level of 0.1 f/cc. While it is theoretically possible to develop mesothelioma from ambient air concentrations, it has not been proven to occur at levels at or below ambient air concentrations. Given that there is no truly "unexposed" population, it would be impossible to reasonably perform such a study to determine if this were the case.

State of the Art:

In 1898 Montague Murray described interstitial fibrosis in an individual exposed to asbestos. Pancoast described radiographic changes of interstitial fibrosis in asbestos workers in 1917. Cooke described two cases of asbestosis in the 1920s, and actually used the term "asbestosis" to describe the interstitial fibrosis among asbestos workers, and also noted pleural plaques (fibrosis) in these workers.

In 1930 Merewether and Price, in their *Report on the Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry*, noted that inhaling dust containing asbestos fibers could lead to disabling and fatal lung disease. They studies asbestos workers in the textile mills in Great Britain, and noted that asbestosis could occur in large numbers of exposed individuals. Moreover, they found that the textile workers with the highest exposures had more asbestosis than workers in areas where asbestos exposure was lower. Merewether and Price noted that asbestos was a potential hazard to health in any industry where dry asbestos products were abraded or otherwise manipulated to generate dust, such as thermal insulating. They recommended warning, education and training of all those individuals who were exposed to asbestos.

Lynch and Smith noted a case of lung cancer in an asbestos worker from South Carolina in 1935. Textbooks in the 1930s, such as A.J. Lanza's textbook on dust disease, included asbestosis as a disease of concern. In 1943, the first case of mesothelioma was associated with asbestos exposure was published by Wedler in Germany. Also in 1943, Hueper from the United States Publich Health Service stated that he believed asbestos caused lung cancer. He published an editorial stating this association in the Journal of the American Medical Association in 1949.

In 1955, Doll published a seminal article that described the increased risk of lung cancer among asbestos exposed workers. By the time of Doll's epidemiology study, there had been over 60 cases of asbestos-related lung cancer published in the literature. In 1960, Wagner et.al. published a study of 33 cases of malignant mesothelioma among individuals who were exposed to asbestos in and around the crocidolite mines in South

Africa. Not only were miners developing disease, but family members, individuals on the wagon routes in which the asbestos was carried and people who had played with mine tailings as children developed mesothelioma. In the early 1960s numerous studies in several countries, under different exposure scenarios, were published that showed mesothelioma in association with asbestos exposure. In fact by the end of 1964, over 700 scientific articles had been published that showed the adverse health effects of asbestos.

The Development of Diseases: When asbestos is inhaled, some proportion of the fibers can be deposited upon any component of the respiratory tract, including the nose, pharynx, conducting airways and the alveolar or gas exchanging regions of the lung. Fibers that land initially on the airways and above are cleared rapidly from the lung. The primary defense mechanism that mediated this clearance is known as the mucociliary escalator. The escalator is comprised of collated and mucous producing epithelial cells that propel inhaled fibers up to the mouth where they can be swallowed or expectorated. These epithelial lining cells are the "target cells" for cancers. Fibers that evade the mucociliary escalator can penetrate into the lower airways and lung tissue, where they can be transported through the body. Amphibole fibers tend to clear from the lung less rapidly than chrysotile fibers. Asbestos is cleared through the pulmonary lymphatics to lymph nodes and to the pleura, the target organ for pleural mesothelioma. Of the different fiber types, Suzuki, Sebastien and LeBouffant have all shown that chrysotile fibers preferentially translocate to the pleural space.

Asbestosis: The fibers that are inhaled and deposited past the escalator can cause asbestosis. These fibers deposit initially on the Type 1 and Type 2 alveolar epithelial cells. On the epithelial surfaces, some asbestos fibers activate the 5th complement which attracts inflammatory cells, including foreign particles, like asbestos, from the lung. About 20% of the fibers deposited on the alveolar surfaces are enveloped by the Type 1 cells and are translocated to the underlying connective tissue (interstitial) compartment. There, the fibers can interact with interstitial fibroblasts, myofibroblasts and macrophages. Fibroblasts and myofibroblasts are the target cells for asbestos because these are the cells that synthesize and release the scar tissue matrix. (See Y. Suzuki & N. Kohyama, *Translocation of Inhaled Asbestos Fibers from the Lung to Other Tissues.*,19 Am J. Indus, Med. 701 (1990); Y. Suzuki & N. Kohyama, *Translocation of Inhaled Asbestos Fibers from the Lung to Other Tissues*., *19* Am J. Indus, Med. 701 (1991)). They produce scar tissue when the epithelial cells are injured and when the macrophages are activated. Alveolar cells and macrophages release a number of protein growth factors that stimulate the fibroblasts to multiply and produce scar tissue, and the fibroblasts and myofibroblasts also synthesize a similar array of factors that induce their own cell growth and matrix production that we recognize as asbestosis. Like *all* of the asbestos-related diseases, asbestosis is dose dependent. An individual typically needs long-term occupational exposure to develop clinical asbestosis.

The scarring process described above begins as soon as inhaled fibers are deposited on the alveolar surfaces, and microscopic asbestosis is ongoing in the lungs of afflicted individuals for many years before any clinical signs or symptoms are presented. The initial physiological symptom of asbestosis is shortness of breath. This is caused by

the scar tissue which replaces normal elastic connective tissue, this producing a stiff lung that restricts the individual from taking a deep breath. Shortness of breath also results when scar tissue thickens the alveolar-capillary membrane, the barrier across which oxygen and carbon dioxide gases are exchanged.

Pleural Plaques and Fibrosis: This is scar tissue formation in an identical manner to that described above, under asbestosis. The difference is that there is little direct deposition of asbestos fibers in the pleura. While some fibers can be inhaled through the alveolar ducts and reach the pleura directly, most fibers that land on alveolar surfaces and reach the interstitial compartment have direct access to the pleura do so by way of pulmonary lymphatic flow. The inhaled fibers that land on alveolar surfaces and reach the interstitial compartment have direct access to lymphatic fluids which flow through these regions on the way to the pleura. The lymphatic flow carries fibers to the pleura where they interact with the sub-mesothelial fibroblasts that produce a scar tissue matrix, as described above. If the scarring is in a circumscribed pattern, the scarring is called "plaque". Investigators have shown that this injury can result in a restrictive lung disease in some individuals.

Lung Cancer: These tumors caused by asbestos typically arise in cigarette smokers, although some epidemiologic studies on asbestos-exposed non-smokers show an increased risk of developing the disease. When an individual is exposed to the cancer-causing agents (carcinogens) of both cigarettes and asbestos, the risk of getting lung cancer is increased well beyond the risk presented by exposure to either agent alone or by simply adding the risks of the two carcinogens. Epidemiologists multiply the risks of the two carcinogens since there is a clear synergy in the way asbestos and cigarette smoke combine to cause lung cancer.

Cancer is the loss of control of cell growth. Every cell in the bodies of humans and animals is under strict genetic control of the rate at which a given cell replaces itself by dividing. Cancer is caused when the specific genes that control cell division and other aspects of the cell cycle develop errors or mutations. Carcinogens induce such errors, and complete carcinogens can produce the errors with no other agent required. Cigarette smoke has a number of complete carcinogens, and all of the asbestos varieties have been shown to act as complete carcinogens. Thus, as the airway epithelial cells of the mucociliary escalator are assaulted daily by cigarette smoke and asbestos fibers, a number of cells are injured, and many exhibit genetic errors through the lifespan of the individual. In those who are susceptible to developing a cancer, one of those injured cells accumulates a sufficient number of genetic errors in genes that control cell growth to finally, after decades of exposure, lose the normal growth pattern and grow into a malignant tumor. (See Frost G, Darton A, Harding AH. *The effect of smoking on the risk of lung cancer mortality for asbestos workers in Great Britain (1971-2005)* Ann Occup Hyg 55:239-24 (2011)).

Mesothelioma: This cancer occurs when a mesothelial cell of the pleural or peritoneal surfaces develops a sufficient number of genetic errors in a set of genes that control cell growth, as described above. Cigarette smoking has no influence on the development of mesothelioma. (See N.S. Offermans, et. al., *Occupational Asbestos Exposure and Risk of*

Pleural Mesothelioma, Lung Cancer, and Laryngeal Cancer in the Prospective Netherland Cohort Study, 56 J. Occupational Envt'l Med. 1 (2014); Robinson BM. *Malignant pleural mesothelioma: an epidemiological perspective,* 1 Annals Cardiothoracic Surgery 491 (2012)).

Asbestos exposure is the only known occupational and/or environmental cause of mesothelioma in North America, and all of the asbestos varieties induce the genetic errors described above and cause this cancer. The fibers that cause mesothelioma reach the pleural surfaces through the lymphatic pathways, as explained earlier, and they interact with the target cells of the mesothelial surfaces. When a sufficient number of genetic errors have accumulated in a single mesothelial cell, this cell can undergo neoplastic transformation and grow into a deadly tumor. It typically takes many decades for a sufficient number of mutations to occur in a single mesothelial cell because of the numerous effective defense mechanisms that destroy genetically defective cells, thus explaining the long latencies known for this cancer.

All of the asbestos varieties have been shown to cause genetic errors and fibers less than five microns can bind DNA and this contributes to the development of genetic damage. Short fibers have been found to accumulate in the pleural regions of the lung as well as in mesenteric lymph nodes of the peritoneal cavity. Longer fibers may be comparatively more dangerous than short fibers (on a fiber per fiber basis), but all size ranges are capable of causing and contributing to the development of mesothelioma or any of the asbestos-related diseases. Exposure to asbestos fibers of all types and lengths are toxic, and short fibers more readily reach the mesothelial target cells of the pleura. (See Y. Suzuki & S. R. Yeun, *Asbestos Fibers Contributing to the Induction of Human malignant mesothelioma.*, 982 Annals N.Y. Acad. Sci. (2002); Y. Suzuki, et al. *Short thin asbestos fibers contribute to the development of human malignant mesothelioma: pathological evidence.*, 208 Int'l. J. Hygiene Envtl. Health 201 (2005)). Fibers of all lengths can bind to DNA and cause genetic errors that are required in the causation of cancer such as mesothelioma. Fiber burden studies of mesothelioma patients show a preponderance of chrysotile asbestos within the tumor tissue. Since the target location of mesothelioma is the pleura, the lung burden of asbestos does not reflect the fact that asbestos has moved from the lung to the pleura, where it can cause the mesothelioma to develop. (See Ronald F. Dodson, *Analysis and Relevance of Asbestos Burden in Tissue, in Asbestos: Risk Assessment, Epidemiology and Health Effects.* Risk Assessment, Epidemiology and Health Effects 78 (2d, ed. 2011); M. Silverstein, et al., *Developments in Asbestos Cancer Risk Assessment.* Am J. of Indus. Med. (2009)).

Moreover, there is ample evidence to support the conclusion that exposure to all types of asbestos fibers, including those found in talc and talcum powder products can and does cause mesothelioma. This conclusion is supported by, among others, the American Conference of Governmental Industrial Hygienists, the American Thoracic Society, the Environmental Protection Agency, the International Agency for Research on Cancer, the National Toxicology Program, OSHA, the Consumer Products Safety Commission, the World Health Organization, and the World Trade Organization. The scientific consensus that all fiber types and sized can cause mesothelioma is also reflected

in the Consensus Report of the 1997 Helsinki Conference (discussed below) and publications from the American Cancer Society and the National Cancer Institute of the National Institutes of Health.

In essence, there exists a consensus among the overwhelming majority of medical and scientific professionals and organizations that asbestos fibers of any type or size can cause mesothelioma, including chrysotile fibers. (See Dodson, Ronald F. et al., *Asbestos Fiber Length as Related to Potential Pathogenicity: A Critical Review,* 44 Am J. Indus. Med. 291 (2003); D. Egilman, et al., *Exposing the "Myth" of ABC, "Anything But Chrysotile: A Critique of the Canadian Asbestos Mining Industry and McGill University Chrysotile Studies.* 44 Am J. Indus. Med. 540 (2003); David S. Egilman & Marion Billings: *Abuse of Epidemiology: Automobile Manufacturers Manufacture a Defense to Asbestos Liability*, 11 Int. J. Occupational Envtl Health 360 (2005). 11:360-371; Egilman D. *Fiber Types, Asbestos Potency, and Environmental Causation.* 15 Int. J. Occupational Envtl. Health (2009); Finkelstein, M. *Asbestos Fiber Concentrations in the Lungs of Brake Workers: Another Look,* 52 Annals Occupational Hygiene 455 (2008); M.M. Finkelstein & C. Meisenkothen, *Malignant Mesothelioma among Employees of a Connecticut Factory that Manufactured Friction Materials Using Chrysotile Asbestos.* 54 Annals Occupational Hygiene 692 (2010); P.J. Landrigan, et al., *The Hazards of Chrysotile Asbestos, a Critical Review*. 37 Indus. Health 271 (1999); W.J. Nicholson, *The Carcinogenicity of Chrysotile Asbestos-A Review*. 39 Indus. Health 57 (2001); R.A. Lemen, *Chrysotile Asbestos as a Cause of Mesothelioma: Application of the Hill Causation Model.* 10 (2) Int. J. Occupational Envtl. Health (2004); *see also* R. Lemen, *Asbestos in Brakes: Exposure and Risk of Disease.* 45 Am. J. Indus. Med 229 (2004); EPA: *Guidance For Preventing Asbestos Disease Among Auto Mechanics.* (1986); A.H. Smith & C.C. Wright, *Chrysotile Asbestos is the Main Cause of Pleural Mesothelioma.* 30 Am. J. Indus. Med. 252 (1996); U.S. Dept. of Labor: *Working Safely with Asbestos in Clutch and Brake Linings.* (posting); U.S. Dept. of Labor, OSHA Directorate of Science, Technology and Medicine, Office of Science and Technology Assessment. *Asbestos-Automotive Brake and Clutch Repair Work*; World Health Organization, *Environmental Health Criteria 203: Chrysotile Asbestos.* International Programme on Chemical Safety (1998 Geneva)).

Asbestos fibers are very small; so small, in fact, that millions of fibers could fill the air in a room without anyone being able to perceive it with the naked eye. The fibers are odorless, cannot be seen with the naked eye, and are aerodynamic. Consequently, someone can inhale asbestos fibers without even being aware of it. The fibers are also small enough to pass through the normal respiratory defense mechanisms that the human body uses to keep out toxins and debris.

The Scientific community has even concluded that small amount of asbestos exposure can cause cancer. The Rodelsperger study indicates that exposure to asbestos below the Occupational Safety and Health Administration (OSHA) Permissible Exposure Level (PEL) of 0.1 fibers per cubic centimeter can cause disease. However, visible asbestos-laden dust that is released into the air from the manipulation of gaskets or packing, or that is reintroduced into the respirable zone from the process of sweeping the

floor, is between 2.0 and 10.0 fibers per cubic centimeter. These levels far exceed the OSHA PEL. Some of these levels even exceed the OSHA PEL issued in 1972.

Government agencies and international organizations universally recognize asbestos as a carcinogen in low levels. These agencies include the International Agency for Research on Cancer, Environmental Protection Agency, OSHA, National Institute for Occupational Safety and Health, and World Health Organization. The inhalation of asbestos fibers also does not trigger any immediate physiological reactions: the victim doesn't experience any immediate irritation, asthmatic problems, or allergic reactions. Moreover, the latency, or development period, for mesothelioma is very long: the minimum latency period is usually considered to be around 10 years with a maximal latency period well over 60 years after the last exposure. Consequently, it could be decades before someone is aware that he or she was exposed to asbestos, or it might have occurred so remotely that they do not realize they had asbestos exposure. Moreover, they may not realize that a product they used contained asbestos and thus are unaware they had exposure.

The Helsinki Criteria for Attribution: In January 1997, a conference called "Asbestos, Asbestosis and Cancer" was held in Helsinki, Finland. The conference was convened to establish criteria for diagnosis and attribution of disorders of the lungs and pleura, including mesothelioma. This was a multidisciplinary group of internationally recognized experts, consisting of pathologists, radiologists, occupational and pulmonary physicians, epidemiologists, toxicologists, industrial hygienists, and clinical and laboratory scientists specializing in tissue fiber analysis. Collectively, the members had published over 1,000 articles on asbestos and associated disorders. The conclusions of the conference were developed into a peer-reviewed Consensus Report that established the "Helsinki Criterion". Among the conclusions of the Helsinki Criterion are:

a. That, in general, reliable work histories provide the most practical and useful measures of occupational asbestos exposure; and
b. That even in the absence of other independent evidence of disease (e.g. lung fiber counts exceeding the background range for the lab in question; the presence of radiographic or pathological evidence of asbestos-related tissue injury; histopathologic evidence of abnormal asbestos content), a history of significant occupational, domestic or environmental exposure to asbestos will suffice for attribution of the disease with asbestos exposure.

Moreover, with reference to determining an occupational etiology of mesothelioma, the Helsinki Criterion Consensus Report concluded that:

a. The great majority of mesotheliomas are due to asbestos exposure;
b. Mesothelioma can occur in cases with low asbestos exposures. However, very low background environmental exposures carry only an extremely low risk;

c. About 80% of mesothelioma patients have had some sort of occupational exposure to asbestos (necessitating a carefully obtained and detailed occupational history for proper diagnosis);
d. An occupational history of brief or low-level exposure should be considered sufficient for mesothelioma to be designated as occupationally related;
e. A minimum of 10 years from the first exposure is required to attribute mesothelioma to asbestos exposure (though in most cases, the latency interval is longer);
f. Smoking has no influence on the risk of mesothelioma.

The conclusions of the Helsinki Criterion have since been adopted by, and form the general consensus of, the medical community's positions vis-à-vis mesothelioma and asbestos. (See *Consensus Report, Asbestos, asbestosis and cancer: the Helsinki criteria for diagnosis and attribution*, 23 Scandinavian J. Work Environ Health 311 (1997)). And, given the fact that about 80% of patients with mesothelioma have had some sort of occupational exposure to asbestos,[1] asbestos exposure in the workplace is a prime focus of Occupational Medicine when dealing with mesothelioma patients.

<u>*Mesothelioma is a dose responsive disease:*</u> It is my opinion that Mesothelioma and asbestos related lung cancer are dose responsive diseases in which more substantial exposures directly increases the risk for the development of these cancers. This linear dose-response relationship presented in *Asbestiform Fibers: Non-occupational Health Risks*, published by the National Research Council National Academy of Sciences in 1984, discussed herein, is neither new nor novel and generally accepted in the medical and scientific communities. As per the aforementioned Helsinki criteria, the first question usually asked of a patient diagnosed with mesothelioma, concerns how, when, and where the patient was exposed to asbestos. (*See Consensus Report*, *Asbestos asbestosis and cancer: The Helsinki criteria for diagnosis and attribution.* 23 Scandinavian J. Work Environ Health 311 (1997)). Because of the proven association between asbestos fibers and mesothelioma, proof of significant exposure to asbestos dust is considered to be proof of specific causation. (*See* P. Boffetta, et al., *Health Effects of Asbestos Exposure in Humans: A Quantitative Assessment.* 89 (6) Medicina Del Lavoro, 471 (1998). This causal relationship between exposure to asbestos dust and the development of mesothelioma is so firmly established in the scientific literature that it is accepted as a scientific "fact".

Malignant mesothelioma is, in general, a dose response disease where each and every significant exposure to asbestos-containing dust has been shown to contribute to cause diffuse malignant mesothelioma including pleural mesothelioma (*See also* Newman, et al., *Malignant Mesothelioma Register 1987-1999.* 74 Int'l Arch Env. Health 383 (2001), (concluding that "higher cumulative asbestos-fiber dose leads to the earlier development of mesothelioma)). As each exposure to asbestos contributes to the total amount of asbestos that is inhaled, and, in doing so, reduces the necessary period for

[1] The remaining 20% of mesothelioma patient likely had asbestos exposures that were para-occupational or are simply unidentified.

asbestos disease to develop. Therefore, each non-trivial exposure to asbestos should be considered a substantial contributing factor in the development of the malignant mesothelioma or lung cancer.

Exposure to Asbestos contaminated talc and disease

Asbestos fibers have been reported in cosmetic talcum powder for decades, in company documents, the media, FDA communications, trade organization documents and the published medical and scientific literature. Cosmetic talc has been analyzed by researchers in various countries, and has routinely been shown to be contaminated with asbestos. Exposure to asbestos contaminated talc has been shown, Cralley, et.al., in 1968 identified the association of cosmetic talc and its potential for causing asbestos related diseases, such as mesothelioma. Case reports identified asbestos from talc as the sole source of exposure in two individuals who developed mesothelioma (see Andrion & Fujiwara). In 1976 Rohl and Langer tested 20 consumer products that had been labeled as talc or talcum powder, including body powders. Of the 20 products that were tested, ten were found to contain tremolite and anthophyllite, principally asbestiform. Of note, the product that had the highest asbestos content in the Rohl and Langer study was the same product later tested by Gordon, et.al. This product was in fact, Cashmere Bouquet.

A recent paper by Gordon, et.al., Asbestos in Commercial Cosmetic Talcum Powder as a Cause of Mesothelioma in Women, evaluated the mineralogical constituents of Cashmere Bouquet and its ability to release asbestos fibers into the breathing zone of the direct user and bystanders. In their paper, Gordon et.al. noted that the talc that was used in Cashmere Bouquet was derived from three distinct regions, where anthophyllite and tremolite asbestos were found. Gordon et.al. measured 18 million anthophyllite asbestos fibers per gram in the talcum powder. Air measurements were done by both phase contrast microscopy (PCM) and transmission electron microscopy (TEM), and significant levels of asbestos fibers were noted (anthophyllite, tremolite and some chrysotile) in the breathing zone of the individual applying the powder as well as a bystander. Results taken from the experiment in the paper show that personal measurements from the shaker container test showed a measurement by PCM of 4.8 f/cc, with an actual asbestos fiber measurement of 1.8 f/cc. Bystander measurements showed a lower, but still significant exposure of 1.35 f/cc by PCM for the bystander, and 0.5 f/cc of actual asbestos fibers. Similar measurements were done with the puff application method. Personal measurements after using a puff were 23.6 f/cc and 16.5 f/cc for the user, with actual asbestos fiber measurements of 5 f/cc and 3.5 f/cc. A short term sample showed even higher measurements, of 60 f/cc with the use of a puff and actual asbestos fiber measurements of 13 f/cc. Bystander exposures to asbestos from the puff application were elevated, with a short term sample by PCM of 13.7 f/cc and 9.7 f/cc, and an actual asbestos fiber measurement of 4.9 f/cc and 3.5 f/cc. Gordon et.al. also noted that the TEM measurements were far more sensitive than x-ray diffraction detection, since there was a much lower detection limit with TEM.

In addition to looking at bulk and air samples, Gordon et.al analyzed the lung tissue and lymph node tissue of a woman who had been exposed to Cashmere Bouquet.

The authors found that there were 3150 and 4150 fibers per gram wet weight, respectively, with a detection limit of 690 fibers per gram wet weight. All fibers were 5 micrometers or greater in length, and had an aspect ratio of 20:1 or greater. The fibers were identified as anthophyllite or tremolite. In addition to the fibers counted above, there were many anthophyllite and tremolite fibers that were less than 5 micrometers in length, with a predominance of anthophyllite. In the lymph node, amphibole asbestos fibers were also noted, measuring 12,738 fibers per gram wet weight (detection limit 2123 fibers per gram wet weight). Again, the fibers noted were anthophyllite and tremolite. In addition to the asbestos found in the lungs, the authors noted fibrous and platy talc and small asbestos bodies.

The issue of asbestos and talc has been studied for decades among talc miners and millers. Lung scarring was seen in miners from New York State in the 1950s, and there are elevated rates of mesothelioma and lung cancer in miners at the asbestos contaminated talc mines (see also Kleinfeld 1967 and Finkelstein 2013). Recently, mesotheliomas in Italian chrysotile miners and millers have been attributed to tremolite asbestos-containing talc near and around that geologic formation (see Ilgren 2015). The International Agency for Research on Cancer has noted that talc contaminated with asbestos is carcinogenic.

Applying an Accepted Method for Evaluating Disease Causation in an Individual

In deciding whether Ms. Dalis's mesothelioma was caused by her exposure to asbestos, I applied the methodology that was described by Welch, et.al. in her paper Asbestos Exposure Causes Mesothelioma, but Not This Asbestos Exposure: An Amicus Brief to the Michigan Supreme Court, published in 2007 in the International Journal of Occupational and Environmental Health. In this paper, she identifies four questions that should be examined in the causation of disease in an individual:

1. Was the individual exposed to a toxic agent?
2. Does the agent cause the disease present in the individual?
3. Was the individual exposed to this substance at a level where the disease has occurred in other settings?
4. Have other competing explanations for the disease been excluded?

For question #2, there is ample literature that asbestos causes mesothelioma and no dispute in the medical literature. Ms. Dalis has no other competing explanations (#4) for the development of her left sided mesothelioma. With respect to question #1, Ms. Dalis has an exposure to asbestos from talcum powder for decades, fulfilling this criterion. Moreover, in a sampling of Ms. Dalis's peritoneal tissue, chrysotile type asbestos (both fibers and fibrils), as well as talc were found in tissue analyzed by Dr. Gordon, demonstrating the prior exposure to asbestos containing talcum powder. Mr. Fitzgerald's analysis of both Cashmere Bouquet and Mennen talcum powders showed asbestos fibers, and showed that they were present in the air after the type of use which Ms. Dalis described. The remaining criterion, #3 is whether there is an analogous exposure scenario in which others also developed mesothelioma. As described above in

the medical and scientific literature, as well as recently published by Gordon, et.al, there are other women and men with exposure to cosmetic talcum powder products and the source talcs who then developed malignant mesothelioma.

Summary and Specific Causation in Ms. Dalis's Case

Based on the information that was provided to me, and applying both my understanding of the medical literature and the facts of this case, it is my opinion to a reasonable degree of medical certainty that the exposures to the dust from asbestos-contaminated cosmetic talc products that Ms. Dalis used for in at least three decades, starting nearly 60 years ago, were above normal background levels. Her exposure to asbestos-contaminated talcum powder was the cause of her mesothelioma. If she had not used asbestos-containing talcum powder, she would not have developed malignant mesothelioma.

The opinions related to Ms. Dalis's case are based on my review of the evidence of exposure in this case, the medical and scientific literature as described above regarding asbestos exposure and disease, pathological studies of Ms. Dalis's periteoneal tissue, available studies concerning fiber release and measurements of asbestos in the specific brands of talcum powder Ms. Dalis used, epidemiological studies of exposure to asbestos and the development of disease, and my knowledge, skill, experience, and training as a physician specializing in occupational medicine with a clinical focus on evaluating individuals with asbestos exposure.

I have attached a partial reference list as Appendix C that indicates reliance materials for this report.

Sincerely,

MD

Jacqueline Moline, MD, MSc, FACP, FACOEM